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Obstetrics &

Study of Morphological and Histopathological Changes in Placenta in Preeclampsia and its Association with Maternal and Fetal Outcome

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Abstract: Hypertensive disorders in pregnancy forms one part of the deadly triad, along with haemorrhage and infection that contribute greatly to maternal and fetal morbidity and mortality. Careful examination of placenta can give information that may be important in the immediate and later management of mother and infant. Microscopic examination of the placenta is important to determine the nature of the pathology, but many disorders show similar features. The study was done in 100 placentas, out of the 100 placentas collected, 50 placentas from uncomplicated full term normal deliveries and another 50 were collected from pre-eclamptic patients. Gross and microscopic examination of the placenta was carried out. The size, shape, weight, thickness at centre, number of cotyledons, and site of insertion of umbilical cord was noted. Pre-eclampsia (PIH) significantly affects the placenta by reducing its weight and dimensions.. It induces histological changes such as, areas of syncytial knot formation, fibrinoid necrosis, calcified areas, hyalinised areas, and areas of medial coat proliferation of the blood vessels.

Keywords: fetal outcome, histopathology and morphology, placenta, pre-eclampsia.

INTRODUCTION

Hypertensive disorders in pregnancy forms one part of the deadly triad, along with haemorrhage and infection that contribute greatly to maternal and fetal morbidity and mortality. Pre-eclampsia [1] is recognized as a specific disorder since the time of Hippocrates, the only completely successful therapy remains delivery of the placenta.

In normal pregnancies, the wall of the spiral arteries is invaded by trophoblastic cells and transformed into large, tortuous channels that carry a large amount of blood to the intervillous space and are resistant to the effects of vasomotor agents. These physiologic changes are restricted in patients with preeclampsia. The main feature of abnormal placentation is inadequate trophoblastic invasion of the maternal spiral arteries [2]. This results in persistence of muscular and elastic tissues of the media of spiral arteries. Babies born with a disproportional large placenta are at a greater risk of developing hypertension in later life [3]. Careful examination of placenta can give information that may be important in the immediate and later management of mother and infant [4]. Microscopic examination of the placenta is important to determine the nature of the pathology, but many disorders show similar features.

MATERIALS AND METHODS

The study was conducted in Department of Obstetrics and Gynaecology in DVVPF's Medical College and Hospital, Ahmednagar during the period of 6 months from date of approval.

The study was done in 100 placentas, out of the 100 placentas collected, 50 placentae from uncomplicated full term normal deliveries and another 50 placentae were collected from preeclamptic patients.

The age of the women varied from 18 to 35 years. The cases will be divided into normal and preeclampsia group.

GROUP A: This group comprises pregnant women without preeclampsia. (n=50)

GROUP B: This group comprises pregnant women with preeclampsia. (n=50)

The placenta with attached membranes and umbilical cord was collected soon after delivery, washed in running tap water, labelled, and then fix with 10% formalin for 4-6 weeks. Gross and microscopic examination of the placenta was carried out. The size, shape, weight, thickness at centre, number of cotyledons, and site of insertion of umbilical cord was noted. The birth weights of newborn babies were documented and feto-placental weight ratio was calculated. Histo-pathological study of placenta was done and the slides were studied under light microscope.

MORPHOLOGICAL STUDY

Shape of placenta

The shape of the placenta and presence of accessory lobe were recorded after proper inspection. Each placenta was categorized as oval, circular or irregular in shape

Diameter

The placenta was placed in a flat tray after trimming and mopping. At first, the maximum diameter was measured with a metallic scale graduated in centimetres (cm). Then a second maximum diameter was taken at right angles to the first one. The mean of two measurements was considered as the diameter of the placenta expressed in centimetre.

Thickness

With a long needle placental thickness was measured at five points of each placenta. Each placenta was placed on fetal surface. The placenta was divided arbitrarily into three zones of equal parts by drawing two circles on the maternal surface. These circles cut the radius of the placenta into three equal parts. One thickness was measured from the centre of the central zone, two from middle and two from peripheral zone. The peripheral points were taken within the outer zone on a line perpendicular to the previous imaginary line. Finally the mean of all five measurements was calculated and considered as thickness of the placenta

Number of Cotyledons

Each formalin-fixed placenta was taken on both hands. Then gentle pressure was applied on the central part of the fetal surface with thumbs of both hands while holding the periphery of the placenta with the other fingers. As a result, the cotyledons on the maternal aspect become prominent after separation between them. Then the placenta was put on a flat tray with maternal side facing upward by placing a block of paraffin on the fetal side. Then counting was started from the left side of the one end of the placenta going rightward and again turning back to the left in a manner of loop. This counting procedure was repeated until the other end of the placenta was reached. The total number of cotyledons was recorded.

HISTOPATHOLOGICAL STUDY

Tissue bits were taken from the implantation of the umbilical cord, marginal sections as 12, 3, 6, 9'o clock positions and center of the placenta, umbilical cord at the placental junction, and cut end and membranes. Additional placental sections are also taken if there is a presence of any fibrosis or infarct. Tissue bits were processed and stained with routine hematoxylin and eosin stain and special stains, i.e., periodic acid-Schiff, reticulin, wherever required. Microscopic sections were studied by arbitrary criteria considering the area of histopathological sections.

The new born babies were inspected for congenital anomalies. Apgar score and birth weights were noted, and fetoplacental weight ratio was calculated in each case.

The data collected from morphological and morphometric studies were recorded. Descriptive statistics was used to analyze the data. They were represented as Mean \pm SD (standard deviation). The statistical significance between the means of the control group and study groups were analyzed by using Students unpaired "*t*" test. A *P* value of <0.05 was considered statistically significant.

OBSERVATION AND RESULTS

Table-1: Shape of Flacenta							
Shape Of Placenta	GROUP A (N=50)	GROUP B (N=50)	P VALUE				
Oval	24	22	>0.05				
Circular	18	17	>0.05				
Irregular	08	11	>0.05				

Table-1: Shape of Placenta

Shape of the placenta: In the present study, in group A and group B, the number of oval shaped placenta was 24 (48.0%) and 22 (44.0%), the number of circular shaped placenta was 18 (36.0%) and 17 (34.0%), the number of irregular shaped placenta was 08 (16.0%) and 11 (22.0%) respectively.(Table-1)

Weight of placenta

In our study, the mean (\pm SD) weight of the placenta was 496 \pm 6.4 in group A and 365 \pm 5.82 in group B. So, it was evident that the diameter of the placenta was statistically different from each other at P<0.05(Table-2).

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Diameter

In the present study the mean (\pm SD) diameter of the placenta was 20.45 \pm 4.12 cm in group A and

 18.86 ± 3.62 cm in group B. So, it was evident that the diameter of the placenta was statistically different from each other at P< 0.05 (Table-2).

Placental Parameters	GROUP A	GROUP B	P VALUE
Weight Of Placenta	496±6.4	365 ± 5.82	< 0.05
Diameter	20.45±4.12	18.86 ± 3.62	< 0.05
Thickness	1.86±0.52	1.48 ± 0.49	>0.05
No. Of Cotyledon	17.20±2.4	16.14±2.02	< 0.05
Fetoplacental Weight Ratio	6.231:1	5.002:1	

Thickness

The mean $(\pm$ SD) thickness of the placenta was 1.86 \pm 0.52 cm in group A and 1.48 \pm 0.49 cm ingroup B. Statistically the difference between groups A and B was not significant (Table-2).

Number of cotyledons

In the present study, the mean (\pm SD) number of cotyledon in group A and B was 17.20 \pm 2.40 and

16.14 \pm 2.02 respectively. Statistical analysis between groups A and B was significant at P<0.05(Table-2)

The mean fetal weight to placental weight ratio was 6.231:1 in group A and 5.002:1 in group B. There is a significant negative correlation between fetal weight to placental weight ratio and PIH severity (spearman's rho = -0.659, p<0.001) i.e. as the PIH severity increases, the fetal weight to placental weight ratio decreases.

Histopathological Findings	GROUP A	GROUP B
Cytotrophoblastic Proliferation In Villi		
<20%	42	19
>20%	08	31
Syncytial Knots		
<30%	38	14
>30%	12	36
Basement Membrane Thickening Of Villi		
<3%	46	39
>3%	04	11
Fibrinoid Necrosis		
<3%	43	21
>3%	07	29
Calcification	09	16
Infarcts	07	21
Retroplacental Clots	04	11
Chorangioma	02	12

 Table-3: Histopathological Findings

On microscopy, cytotrophoblastic proliferation (>20%) was seen in 08(16%) of Control group and in group B 31 (62.00%) cases with cytotrophoblastic proliferation of >20%, while 42 (84.0%) cases showed cytotrophoblastic proliferation of <20% in group A and 19(38.0%) cases in group B.

Excess cytotrophoblastic proliferation is directly proportional to the severity of the disease. Placental cytotrophoblastic proliferation of <20% was more in control group than in PIH. These findings were statistically significant (Table-3).

Control group showed 38(76.0%) placentae with <30% syncytial knots and 12 (24.0\%) placentae with >30% syncytial knots. In group A, 14(28.0\%) cases showed presence of syncytial knots in <30% of

villi, whereas 36(72.0%) cases showed syncytial knots in>30% of villi (Table-3).

Group A, 4(8.0%) showed >3% basement membrane thickening, while 46(92.0%) had <3% basement membrane thickening, 11(22.0%) of Group B showed presence of basement membrane thickening in >3% of villi while 39 (78.0%) placentae had Basement membrane thickening in <3% of villi. These findings were statistically significant (Table-3).

07(14.0%) of group A showed fibrinoid necrosis in >3% villi and 29 (58.0%) cases had fibrinoid necrosis in >3% villi. Fibrinoid necrosis in <3% of villi was seen in21 (42.0%) cases of group B and in 43 (86.0%) patients of controlgroup. These findings were statistically significant [Table 3].

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Calcification was present in 09(18%) of group A and 16(32.0%) of group B in our study. 7(14%) of group A and 21(42%) of group B cases showed infarcts (Table-3).

In our study, retroplacental clots was present in 4(8.0%) of group A and 11(22.0%) of group B. Chorangioma a benign placental tumour present in 2(4%) placentae of group A and 12(24%) placentae of preeclampsia group (Table-3).

Table-4. Fetal outcome in two groups				
	GROUP A	GROUP B		
LBW (<2.5 KG)	8 (16%)	34 (68%)		
PERINATAL LOSS	00 (0%)	00 (0%)		
LOW APGAR SCORE	03 (6%)	15 (30%)		

Table-4: Fetal outcome in two groups

In group A, there was no perinatal loss; however, 08(16%) had low birth weight babies and 3(6%) had low Apgar score.

In cases of pre-eclampsia (group B), there was no perinatal loss; however, 34(68%) had low birth weight babies and 15(30%) had low Apgar score [Table-4].

DISCUSSION

Normally a placenta weighs from 450gms to 500 gms. In the present study, the mean placental weight was 496 gms and in PIH it was 364. A significant negative correlation was calculated between the placental weight and severity of PIH. Fox [5] have shown that placentae tend to be smaller in preeclampsia than those in uncomplicated pregnancies.

In the present study, shape of the most placentae were oval (44%) in preeclampsia group and most being circular (48%) in control group. Shah [6] found no clinical significance in oval or round shaped placentae. Irregular shaped placentae were seen in prematurity due to toxaemia.

Cibils [7] reported that the placentae from hypertensive patients were significantly smaller than the normal suggesting that the pathologic process interferes with the normal placental growth.

Majumdar [8] and Kurdukar *et al.* [9] observed that fetal birth weights were lower in cases of preeclampsia; our findings correlated with this studies. Chakravorty [10] also noted similar findings of 5.8:1, which correspond to the findings in the present study.

Dutta and Dutta [11] found calcification in 4 out of 32 (12.5%) cases of normal pregnancy and 26 (44.3%) placentae from PIH group of 59 cases. In our study in GROUP A out of 50 placenta calcification was found in 9 i.e 18% were as in GROUP B 16 placentae(32%) were found calcified. This finding was consistent with the Dutta and Dutta study.

In the present study, cytotrophoblastic proliferation observed was comparable to the study

done by Kurdukar *et al.* The presence of >20% cytotrophoblastic proliferation was associated with low Apgar score in GROUP B was found to be 15 (30%) and in GROUP AN only 3 babies having low Apgar score i.e (6%).

In a study done by Avasthi and Micha[12] found an increase in the knot count with 84% cases of severe PIH and 100% cases of eclampsia, while in the present study, we found all the cases of severe PIH and eclampsia showing syncytial knot count more than 30% on low power view.

Microscopic findings of localized fibrinoid necrosis, endothelial proliferation of arteries, and hyalinization depict the mosaicism of placenta and probably the aftermath of hypertension Teasdale [13] and Udainia *et al.*[14].

According to Bandana Das *et al.* [15] al in 1996, the presence of Retroplacental hematoma was associated with low Apgar score babies larger hematoma was also associated with intrauterine fetal death, because a considerable portion of the villi are acutely separated from the maternal utero placental circulation.

Chorangioma is a rare benign tumour of placenta found on a fetal surface of placenta. In our study this tumour was found in 2 placentae in Group A and in GROUP B 12 placentae were associated with this tumour. This tumour may be associated with pregnancy complication as suggested by the study P Kuhnel [16].

CONCLUSION

Preeclampsia (PIH) significantly affects the placenta by reducing its weight and dimensions. However, it does not have any effect on placental shape, umbilical cord insertion, and number of cotyledons on maternal surface. It induces histological changes such as, areas of syncytial knot formation, fibrinoid necrosis, calcified areas, hyalinised areas, and areas of medial coat proliferation of the blood vessels. These changes compromise utero-placental blood flow and significantly reduce the neonatal birth weight.

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